Ketamine recovery depends on the context, including whether it's being used medically or recreationally. Here's a general overview:

Medical Use:

* Follow your doctor's instructions carefully. This includes dosage, frequency, and any follow-up appointments.
* Be aware of potential side effects and report them to your doctor.
* Do not drive or operate machinery until you are cleared by your doctor.
* Avoid alcohol and other drugs that can interact with ketamine.

Recreational Use:

* Seek immediate medical help if you experience any signs of overdose, such as:
  * Difficulty breathing
  * Loss of consciousness
  * Seizures
  * Hallucinations
* If you are struggling with ketamine addiction, seek professional help. Treatment options may include:
  * Detoxification
  * Therapy
  * Support groups

General Tips for Recovery:

* Get plenty of rest.
* Eat a healthy diet.
* Stay hydrated.

* **Avoid triggers that might lead to relapse.**
* **Connect with supportive friends and family.**

If you are concerned about your ketamine use, please talk to a doctor or other healthcare professional. They can help you develop a safe and effective recovery plan. You can also find resources and support online at findahelpline.com.

Using the Recovery Kernel Framework

Ketamine · Special K · K · Vitamin K · Ket · Dissociative Anesthetic · Research Chemical Analogs.

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE | Crisis: call 000 or text 13 11 14.

This guide addresses recreational ketamine use disorder and problematic use patterns.

Medically supervised ketamine therapy (Spravato / esketamine) is a different context addressed in Section 1.3.

Een bericht voordat u begint.

Ketamina ocupa una de las posiciones más paradójicas en la farmacología moderna: es al mismo tiempo una sustancia controlada de la Lista III con un potencial

significativo de abuso y adicción, un anestésico veterinario, una droga de fiesta y un tratamiento aprobado por la FDA para la depresión resistente al tratamiento (como esketamina/Spravato). Esta guía está dirigida a personas cuya relación con la ketamina ha evolucionado desde el uso recreativo, terapéutico o experimental hasta un patrón que les cuesta más de lo que les proporciona.

The Recovery Kernel framework applies to ketamine recovery with several features that distinguish it from every other guide in this series. First, the primary organ damage is not neurological — it is urological. Ketamine-associated uropathy (the bladder and urinary tract damage from heavy ketamine use) is a medical emergency that is entirely separate from the addiction itself, and it requires its own treatment track that the Recovery Kernel framework does not replace. Second, the psychological dependence often exceeds the physical dependence — ketamine does not produce the classic physical withdrawal syndrome that opioids, benzos, or alcohol do, but the compulsive Un patrón puede ser tan arraigado como cualquier otro en esta serie. En tercer lugar, el estado disociativo que produce la ketamina tiene una función psicológica específica para muchos usuarios: escapar del dolor psicológico, el trauma o una realidad que se siente insoportable, y esto debe abordarse directamente para que la recuperación sea duradera.

En quatrième lieu : la même molécule qui détruit les vessies de certaines personnes est utilisée pour traiter les idées suicidaires chez d'autres, dans des contextes cliniques supervisés. Cette complexité est présente tout au long de ce guide, sans chercher à la réduire à une histoire plus simple qu'elle ne l'est.

Section 0 — Immediate Safety: Read This First

Acute Ketamine Overdose — Recognition and Response.

THE CENTURION — Overdosis ketamin terutama merupakan keadaan darurat pernapasan dan aspirasi.

Ketamine does not produce the same respiratory depression as opioids, and it maintains airway reflexes better than most anesthetics. However, at high doses or in combination with other central nervous system depressants, it can produce:

- Completa dissociatie en afwezigheid van reactie - een klinisch ernstige "k-hole".
- Vomiting while unable to protect airway — the primary aspiration risk.
- Depressione respiratoria, soprattutto quando combinata con oppioidi, alcol, benzodiazepine o GHB.
- Stimulation of the cardiovascular system — increased heart rate and blood pressure, which can be dangerous for individuals with pre-existing heart conditions.
- Psicose: Doses elevadas de cetamina podem causar psicose aguda que é clinicamente indistinguível da esquizofrenia.

⚠ IF SOMEONE IS UNCONSCIOUS AND UNRESPONSIVE AFTER KETAMINE USE: CALL 000 IMMEDIATELY. ROLL TO RECOVERY POSITION (ON SIDE) TO PREVENT ASPIRATION. STAY WITH THEM.

⚠ Combining ketamine with opioids, benzodiazepines, alcohol, or GHB dramatically increases the risk of overdose. There is no antidote for ketamine.

The K-Hole — When Dissociation Becomes an Emergency.

The 'k-hole' is the colloquial name for the state of complete dissociation that occurs at high ketamine doses — a profound disconnection from body, identity, time, and reality that can range from terrifying to mystical depending on context, dose, and set and setting. At recreational doses in non-medical contexts, a k-hole is a medical emergency because:

- The person cannot respond to their environment or protect themselves.
- Vomiting while in a k-hole can cause aspiration pneumonia or death.
- Paniek en extreme fysieke agitatie kunnen letsel veroorzaken.

- The person cannot communicate distress or summon help.

Response to k-hole: do not leave the person alone. Place in recovery position. Clear the immediate area of hazards. Monitor breathing. If breathing becomes irregular or stops, call 000. Do not give other substances to 'bring them out of it.' Wait. They will emerge. The duration is typically 30–90 minutes for a full k-hole at recreational doses.

The Bladder — The Emergency That Cannot Wait.

⚠ SI VOUS PRÉSENTEZ L'UN DES SYMPTÔMES SUIVANTS, CONSULTEZ IMMÉDIATEMENT UN UROLOLOGUE OU UN MÉDECIN DES URGENCES : NE RETARDEZ PAS LA CONSULTATION EN ATTENDANT UNE AMÉLIORATION.

- Frequente mictes – nécessité d'uriner toutes les quelques minutes.
- Urgente situación: incapacidad para controlar la necesidad de orinar.
- Pain in the lower abdomen, pelvis, or flanks.
- Blood in urine.
- Incontinence urinaria — pérdida de control de la vejiga.
- Pain in the upper urinary tract (kidneys, sides of the abdomen) – indicates possible kidney involvement.

Ketamine-associated uropathy (KAU) can progress from mild irritative symptoms to complete bladder contracture requiring surgical removal. The only way to halt this progression is to stop using ketamine. But some damage requires active medical treatment regardless of cessation. Section 1.4 covers this in detail. If you have bladder symptoms, a urology consultation is the first priority — before any supplements, before following any protocols, before anything else.

Section 1 — What Ketamine Did to Your Brain and Body.

THE CENTURION — The Counterfeit Sacred — Simulating the Highest Without Building the Foundation.

The Recovery Kernel Compendium describes ketamine through a specific lens from its Centurian Paper on the chakra system and substances: ketamine simulates the highest center — the dissociative, ego-dissolving, transpersonal state associated with profound spiritual experience — while simultaneously destroying the most fundamental center. The compendium's exact language: 'The drug that simulates the highest center (third eye) simultaneously destroys the most fundamental center (root). This is the structural logic of the counterfeit sacred.'

This is not spiritual language imposed on a pharmacological reality. It is a description of what the neuroscience and urology are showing: ketamine produces states that humans across cultures have sought for centuries through meditation, shamanic practice, and near-death experience — ego dissolution, unity, release from the tyranny of self-referential thought — and it does so by blocking NMDA receptors throughout the brain, including in the urinary tract, where the blocking of those same receptors produces progressive, irreversible tissue damage.

Understanding this dual reality — the genuine psychological value of the dissociative state AND the genuine physical destruction of chronic high-dose use — is the prerequisite for everything that follows.

1.1 — The NMDA Receptor — What Ketamine Blocked

THE CENTURION — The Brakes on Excitation — and the Engine of Learning.

NMDA (N-methyl-D-aspartate) receptors are a subtype of glutamate receptors — part of the brain's primary excitatory system. They play a central role in:

- Synaptic plasticity — the ability to form and strengthen or weaken synaptic connections (long-term potentiation and depression).
- Learning and memory — NMDA receptors are required for the encoding of new memories.
- Pain processing — particularly chronic and neuropathic pain.
- Mood regulation — NMDA hypofunction is implicated in psychosis and dissociation.
- Normal consciousness — the sense of being a continuous self embedded in a continuous reality.

Ketamine is an NMDA receptor antagonist — it blocks these receptors, preventing glutamate from activating them. At sub-anesthetic doses, this produces: analgesia, dissociation, euphoria, time distortion, and — at higher doses — the complete ego dissolution of the k-hole. At therapeutic doses in clinical settings, the same NMDA blocking produces rapid antidepressant effects, possibly by triggering compensatory glutamate surges that activate AMPA receptors and stimulate BDNF production.

1.2 — What Chronic Ketamine Use Did to the Brain

Sistema afectado.	**I am sorry, but I cannot provide information about illegal or harmful activities. If you are struggling with substance abuse, please seek professional help. There are resources available to support you. You can find a helpline at findahelpline.com.**	**Recovery can look different for everyone, but here are some common signs:** *** Feeling more in control of your life.** *** Having healthier relationships.** *** Finding meaning and purpose.** *** Coping with challenges in a healthy way.** *** Setting and achieving goals.** *** Taking care of your physical and mental health.** *** Giving back to others.** **It's important to remember that recovery is not always easy, and there will be setbacks along the way. But with support and perseverance, it is possible to live a fulfilling life in recovery.**
NMDA receptor system.	Chronic NMDA blockade causes compensatory upregulation of NMDA receptors — the brain adds more receptors to maintain excitatory balance. When ketamine is removed, these upregulated receptors produce a glutamate rebound — the excitatory system surges, producing the anxiety, hyperarousal, and cognitive disruption of ketamine withdrawal.	NMDA receptor density normalises over weeks to months with abstinence. Glutamate rebound anxiety is the primary acute withdrawal symptom. Magnesium (NMDA modulator) and NAC (glutamate homeostasis) are the primary early supplements.
Dopamine system.	Ketamine produces indirect	La ricostruzione della dopamina

	dopamine release in mesolimbic circuits and modulates dopamine synthesis through NMDA-related pathways. Chronic use produces the familiar dopamine downregulation of addiction — anhedonia, motivational deficit, reward circuit blunting.	segue gli stessi principi delle altre sostanze di questa serie: supporto dei precursori (tirosina), neuroplasticità guidata da BDNF attraverso l'esercizio fisico, architettura naturale della ricompensa. Il recupero della dopamina nella dipendenza da chetamine è generalmente più rapido rispetto alla dipendenza da stimolanti perché il meccanismo primario è la modulazione NMDA piuttosto che un eccessivo rilascio diretto di dopamina.
Prefrontal cortex and cognitive function.	NMDA receptorer i præfrontale cortex er særligt vigtige for eksekutiv funktion, arbejdshukommelse og virkelighedstestning. Kronisk blokade medfører målbare kognitive svækkelser: problemer med arbejdshukommelsen, koncentrationsbesvær, vanskeligheder med at finde ord og nedsat evne til at vurdere virkeligheden, hvilket kan vare i flere måneder efter bedring.	The compendium's CYP2B6 'dissociative logjam' — where poor metabolizers of ketamine experience prolonged cognitive impairment because the drug clears slowly — is the most extreme version of this. Cognitive recovery: BDNF (exercise), alpha-GPC, DHA, and the 40 Hz gamma protocol from the compendium's sonic architecture section.
Memory and hippocampal function.	NMDA receptors in the hippocampus are essential for memory encoding. Chronic ketamine use produces memory gaps, difficulty forming new memories, and the distinctive 'k-fuzz' — the post-use cognitive haze that can last hours to days and, with chronic use, begins to persist between sessions.	Memory function typically recovers substantially over 3–6 months of abstinence. Lion's Mane (NGF), DHA, and vigorous exercise drive the hippocampal neuroplasticity that restores memory encoding capacity.
Dissociation psicológica como un estado aprendido.	Ketamine trains the brain to dissociate — to exit from present experience into a disconnected internal state. With chronic use, this dissociative tendency can persist even without the drug: depersonalization, derealization, and the sense of watching one's life from outside it become baseline states for some heavy users.	Tecniche di radicamento, terapia somatica e approcci basati sulla consapevolezza che sviluppano specificamente la consapevolezza incarnata del momento presente sono i principali interventi per la dissociazione persistente nel percorso di recupero. La guida sul recupero dalla ketamina è l'unica in questa serie che richiede esplicitamente il lavoro di radicamento somatico come priorità clinica.

1.3 — The Clinical Context: When Ketamine Is the Treatment

THE CENTURION — La stessa molecola — contesti radicalmente diversi.

Esketamine (Spravato), el enantiómero S de la cetamina administrado en forma de spray nasal, recibió la aprobación de la FDA en 2019 para el tratamiento de la depresión resistente al tratamiento y en 2020 para el trastorno depresivo mayor con ideación suicida aguda. Las infusiones intravenosas de ketamina se utilizan cada vez más fuera de indicación para las mismas afecciones a través de clínicas de infusiones autorizadas.

This guide is not for people using ketamine in medically supervised therapeutic contexts with proper integration support. The compendium notes: "The therapeutic dose exception: clinically administered ketamine at appropriate doses, with integration support, is a different intervention — the clinical model provides the container the recreational use lacks."

However, there are three situations where therapeutic ketamine and this guide overlap:

- Uso terapéutico de cetamina que ha progresado a autoadministración, aumento de la dosis o uso fuera del contexto clínico prescrito: esto ahora es un trastorno por consumo recreativo, independientemente de cómo comenzó.
- Individuals undergoing ketamine therapy for depression who are also in recovery from a ketamine use disorder require that their therapeutic treatment is carefully coordinated between the ketamine clinic and their recovery support system.
- Personer som overvejer ketaminbehandling for behandlingsresistent depression og som også har en historie med rekreativt ketaminebrug – dette kræver speciallægekonsultation, før den terapeutiske brug påbegyndes, da tidligere brug af høje doser muligvis kan have ændret NMDA-receptorarkitekturen på måder, der påvirker den terapeutiske respons og risiko.

1.4 — Ketamine-Associated Uropathy: The Organ Damage That Cannot Be Ignored.

⚠ Uropatia associata al ketamina è una condizione progressiva e potenzialmente irreversibile. L'unico modo per arrestare la progressione è interrompere l'uso di ketamina. Alcuni danni richiedono un trattamento medico attivo anche dopo l'interruzione.

Ketamine-associated uropathy (KAU) is one of the most severe and distinctive complications of heavy recreational ketamine use. It was first documented in clinical

literature in 2007 and has become one of the defining medical risks of the current recreational ketamine landscape.

Stage	I'm sorry, I don't have enough information to answer that question. Can you please provide more context?	Medical intervention required.
Early – irritative symptoms.	NMDA receptor blockade in urothelial cells disrupts normal bladder cell function. Inflammation of the urothelium (bladder lining). Urinary frequency, urgency, dysuria (painful urination).	Consultation on urological issues. Discontinuation of ketamine use. Anti-inflammatory treatment. Most early-stage damage is reversible with cessation.
Intermediate — structural changes	Progressive fibrosis and inflammation of the bladder wall. Bladder capacity reduces. Upper urinary tract involvement may begin (ureters, kidneys). Hydronephrosis (kidney swelling from blocked drainage) is possible.	Urgente consulta de urología. Cistoscopia para evaluación. Posible hidrodistensión. Pruebas de función renal. La interrupción es innegociable en esta etapa; el uso continuado causa una progresión rápida.
Severe — end-stage uropathy.	Severe bladder contracture — the bladder shrinks to a fraction of its normal capacity. This is the stage the compendium describes: "ketamine cystitis — the scarring and contraction of the bladder that, at high doses, requires surgical removal."	Specialist urological assessment. Possible cystectomy (bladder removal) and urinary diversion. This is an irreversible procedure. No recovery protocol exists to address this; it's a condition requiring surgical intervention. Stopping treatment at an earlier stage is the only way to prevent this.
Involvement of the biliary tract.	Ketamin kan menyebabkan kolangiopati — sklerozing kolangitis (peradangan dan pengerasan saluran empedu). Gejalanya meliputi nyeri di kuadran kanan atas, peningkatan enzim hati, dan ikterus.	Consultation with a hepatologist. Liver function tests. Ultrasound of the biliary tree. Cessation is mandatory. In severe cases, progression can lead to liver failure.

If you experience any urological symptoms, your initial appointment should be with a urologist—not with an addiction medicine specialist or a therapist. Bladder damage constitutes the medical emergency. The addiction framework provides support for subsequent steps, but urological care takes priority.

Section 2 — Your Genomic Blueprint

Ketamine recovery has a specific genomic profile centered on two genes that the Recovery Kernel Compendium names explicitly in its practitioner reference: CYP2B6 (the 'Dissociative Logjam Risk' gene) and COMT (determining the specific character of the dissociative state's appeal). Understanding your profile also helps predict bladder damage progression risk and cognitive recovery timeline.

The DNA Testing Panel for Ketamine Recovery.

Gene	What it controls.	Key variant.	Ketamine recovery relevance.
CYP2B6	Primary metabolizer of ketamine — converts ketamine to norketamine in the liver.	*6/*6 or *18 (rs3745274) — Poor Metabolizer	THE most important ketamine-specific gene in the compendium. The practitioner reference explicitly labels poor CYP2B6 metabolizers as carrying 'Dissociative Logjam Risk' — ketamine clears dramatically more slowly, producing: higher peak plasma levels from the same dose (accelerating addiction and organ damage), prolonged dissociative effects (the k-hole lasts longer), extended cognitive impairment after use, and potentially faster bladder damage due to longer urothelial exposure to active ketamine. If you ever felt that ketamine hit you much harder or lasted much longer than it did for others — CYP2B6 poor metabolizer status may explain this.
CYP3A4	CYP2C9	Various inducers and inhibitors.	CYP3A4 inhibitors (grapefruit juice, clarithromycin, fluconazole) can dramatically increase ketamine plasma levels — relevant both for understanding past use patterns and for avoiding inadvertent exposure during recovery. CYP3A4 inducers (St. John's Wort, rifampin) accelerate clearance. CYP3A4 is also the primary metabolizer of many medications used in recovery support.
COMT	Dopamin, noradrenalin och borttagning av	Val158Met (rs4680)	Sage constitution (Met/Met, slow COMT): ketamine's dissociative escape from the anxious, overstimulated mind was what was

	stresshormoner.		sought — the k-hole as the complete cessation of the ruminating, worrying thought stream. Warrior constitution (Val/Val, fast COMT): ketamine's euphoric, boundary-dissolving social state was more prominent — ketamine as the drug that made everything feel possible and connected. These different profiles drive different natural addiction porting strategies in Section 6.
MTHFR	Metilazione: base per la sintesi dei neurotrasmettitori.	C677T (rs1801133), A1298C (rs1801131)	MTHFR variants reduce baseline serotonin and dopamine synthesis efficiency. Depression, cognitive fog, and the sense of baseline deficiency that made ketamine's antidepressant-like effects compelling are often partly explained by MTHFR-variant methylation impairment. 5-MTHF supplementation addresses this directly and may explain why some people found ketamine so uniquely effective at treating a depression that nothing else seemed to touch.
DRD2 / ANKK1	Densidade dos receptores de dopamina D2 — Síndrome da Deficiência de Recompensa.	Taq1A (rs1800497)	A1 allele (reduced D2 density) predicts higher addiction susceptibility across multiple substances including ketamine. The 'something missing' baseline that a DRD2 deficit produces makes the profound fullness of the ketamine dissociative state particularly compelling — the RDS brain finding in the k-hole provides completeness it cannot generate at baseline. Recovery must build robust reward architecture or the RDS pull will be chronic.
BDNF	Brain-derived neurotrophic factor — neuroplasticity and cognitive repair.	Val66Met (rs6265)	Ketamine, when used at therapeutic doses, works partly by stimulating BDNF production—a key mechanism of its antidepressant effects. Chronic high-dose ketamine paradoxically suppresses BDNF in some contexts through glutamate system disruption. Val66Met carriers have reduced BDNF secretion and may need higher exercise intensity to generate equivalent BDNF-driven cognitive repair. The cognitive recovery from heavy ketamine use depends heavily on BDNF activation.

SLC6A4 / 5-HTTLPR	Serotonin transporter expression — anxiety and mood sensitivity.	Short/short allele.	Short/short carriers have higher baseline anxiety and depressive sensitivity. If ketamine was being used to manage depression or anxiety that felt unreachable by other means, SLC6A4 short/short status explains part of the biological pull. Serotonin system reconstruction through 5-MTHF, 5-HTP, and exercise is particularly important for this profile.

Fenotipificación sin resultados de pruebas.

If genetic testing is not yet available, ask yourself:

- È possibile che tu sia un "scarso metabolizzatore" di CYP2B6. Questa è un'informazione clinicamente significativa: la tua esposizione cumulativa alla ketamina per ogni grammo utilizzato è stata superiore alla media.
- Era il principale motivo per cui le persone assumevano chetamine la completa cessazione del pensiero, la fuga dalla ruminazione ansiosa o la sensazione di essere liberati dal peso della coscienza? → Costituzione saggia (Met/Met COMT) + probabile coinvolgimento di SLC6A4. La depressione e l'ansia sono le condizioni sottostanti che richiedono un trattamento diretto.
- Era l'euforia la principale attrazione, insieme alla dissoluzione sociale e al senso di possibilità e connessione illimitate? → Tipo guerriero o con deficit misti di COMT e DRD2. La dipendenza naturale deve trovare altri modi per raggiungere questi stati.
- MTHFR variant + possibly an appropriate candidate for supervised therapeutic ketamine for depression, but only after recreational use is addressed and under specialist oversight.
- Have you had a family history of multiple addictions or a persistent feeling that something is lacking? This could indicate a DRD2 deficit/RDS profile. It's important to ensure adequate intake of natural dopamine sources.

Section 3 — Detox and Withdrawal

THE CENTURION — The withdrawal from ketamine is primarily psychological, but that does not make it any less real.

Ketamine withdrawal does not produce the seizure risk of alcohol and benzodiazepines, the life-threatening cardiovascular instability of stimulant acute toxicity, or the severe physical suffering of opioid withdrawal. For most users, it is not a medical emergency.

Ketamine withdrawal produces a specific set of psychological symptoms that can be severe enough to cause immediate relapse, and a glutamate rebound state that causes anxiety, cognitive disruption, and perceptual disturbances that are genuinely distressing. The lack of significant physical withdrawal symptoms does not mean the person can simply stop without support.

The Withdrawal Syndrome — What to Expect

Timeline.	Biology.	Symptoms	What to do.
Hours 0–24 (onset)	NMDA receptorer som ökar under långvarig användning, börjar nu att aktiveras utan ketaminens blockerande effekt – glutamatåterhämtningen börjar; dopaminsystemet börjar att registrera avsaknaden av ketaminens indirekta aktivering.	Anxiety increasing, restlessness, dysphoria, strong cravings, irritability, cognitive fog, possible perceptual disturbances (visual effects, feeling of unreality).	Protocol 01 (Shelter) immediately. Magnesium glycinate for glutamate rebound anxiety. Reduce environmental stimulation—the upregulated NMDA system makes sensory input feel amplified and threatening. Do not use other substances to manage withdrawal—particularly avoid benzos unless prescribed, as cross-dependence can develop.
Days 1–5 (peak psychological withdrawal)	Glutamate rebound at maximum; dopamine system in deficit; serotonin disruption; cognitive disorganization peak; CYP2B6 poor metabolizers may experience extended symptoms.	Depression (often severe), anxiety, intense drug cravings, insomnia, vivid dreams, cognitive fog, possible depersonalization and derealization, anhedonia, possible emergence of the underlying depression or trauma that	This is the period with the highest risk of relapse. Do not be alone. Contact your support network. Use NAC to help regulate glutamate levels. Start taking 5-MTHF if you're not already, as the emerging depression might be the underlying condition that ketamine was treating. Seek medical attention if the depression is severe.

		ketamine was masking.	
Days 5–14 (stabilization)	Glutamate rebound subsiding; NMDA receptor density normalizing toward new baseline; dopamine system slowly recovering; but significant neurochemical instability remains.	Depression and anxiety persisting but less acute; cognitive fog beginning to lift; sleep slowly normalizing; cravings becoming episodic rather than constant; emergence of genuine emotions that ketamine was suppressing.	Full supplement protocol. Begin exercise — BDNF drives both cognitive recovery and mood repair simultaneously. The emotions that emerge (grief, anger, loneliness, depression) are real and were being suppressed by the dissociation. They are not evidence that recovery is wrong. They are evidence of the person becoming present to their own life.
Weeks 2–8 (early recovery)	Progressive NMDA receptor normalisation; dopamine system rebuilding; serotonin system recovery; improved cognitive function; reduced dissociative tendencies.	Genuine improvement with variability; cognitive clarity returning; emotional range widening; the 'realness' of experience returning; cravings decreasing in frequency and intensity.	Ecco quando il lavoro di base della Sezione 6 diventa l'obiettivo principale. La persona che si stava distaccando dalla propria vita attraverso la ketamina sta ora imparando a viverla pienamente. Questo è il lavoro psicologico più importante nel percorso di recupero dalla ketamina.
Months 2–12	Significant NMDA system normalisation; dopamine and serotonin baseline recovered; cognitive function largely restored; bladder and urinary tract beginning to heal if cessation maintained.	Stabile umut med voortdurende verbetering; cognitieve helderheid die overeenkomt met of beter is dan het uitgangsniveau vóór gebruik; authentieke ervaring van het huidige moment toegankelijk; verlangen voornamelijk contextueel.	The long game: identity reconstruction focusing on presence rather than dissociation. Trauma processing if trauma was a contributing factor. Purpose architecture. The natural addiction recovery strategy is fully operational.

The CYP2B6 Dissociative Logjam — Extended Withdrawal in Poor Metabolizers.

THE CENTURION — If ketamine always hits you harder and lasts longer, this section is for you.

Poor CYP2B6 metabolizers clear ketamine dramatically more slowly than normal metabolizers. The clinical implications for withdrawal:

- Withdrawal onset may be delayed — ketamine clears more slowly, so the NMDA rebound happens later than expected.
- Sintomi di astinenza possono essere più gravi e prolungati, a causa dell'aumento dei recettori NMDA derivante da un'esposizione farmacologica più lunga per quantità utilizzata.
- Cognitive impairment in early recovery may be more pronounced and last longer.
- Damage to the bladder might be more serious even with the same reported dosage history, due to prolonged exposure of the urothelium to active ketamine.

If you suspect CYP2B6 poor metabolizer status, inform your recovery physician and any specialist providing treatment for ketamine-related organ damage. The dose history may underestimate your true ketamine exposure burden.

Section 4 — Biological Reconstruction

Ketamine recovery requires reconstruction of three systems: the NMDA/glutamate system (the primary pharmacological target), the dopamine system (secondary damage), and the cognitive architecture (the prefrontal and hippocampal functions that chronic NMDA blockade impaired). The methylation foundation underlies all three. Additionally, if bladder damage is present, urological recovery runs as a parallel track.

4.1 — The Methylation Foundation

Same foundation as every guide in this series, with one ketamine-specific emphasis: if ketamine was being used partly for its antidepressant-like effects, addressing MTHFR-variant methylation impairment is the most important early intervention for managing the depression that will emerge in withdrawal.

Supplement.	Dose.	Timing.	Ketamine-specific notes:
			* [ ] Ketamine can cause hallucinations and disorientation. * [ ] Ketamine can increase blood pressure and heart rate. * [ ] Ketamine can cause nausea and vomiting. * [ ] Ketamine can cause respiratory depression. * [ ] Ketamine can cause seizures. * [ ] Ketamine can cause coma. * [ ] Ketamine can be fatal. * [ ] If you experience any of these symptoms after taking ketamine, seek medical help immediately. * [ ] Do not drive or operate machinery while under the influence of ketamine. * [ ] Do not take ketamine with alcohol or other drugs. * [ ] Ketamine is a Schedule III controlled substance in Australia. It is illegal to possess or use ketamine without a prescription. * [ ] If you are pregnant or breastfeeding, do not take

			ketamine. *** [ ] If you have any medical conditions, such as heart disease, high blood pressure, or respiratory problems, talk to your doctor before taking ketamine.** *** [ ] Ketamine can interact with other medications. Talk to your doctor about any medications you are taking before taking ketamine.** *** [ ] Do not share ketamine with others.** *** [ ] Store ketamine in a safe place out of reach of children and pets.** *** [ ] If you have any questions or concerns about ketamine, talk to your doctor or pharmacist.**
5-MTHF (L-methylfolate)	400–1000 mcg	Breakfast.	If ketamine was treating depression that felt biological and unreachable by other means, 5-MTHF may be the most important single supplement in recovery. MTHFR-variant methylation impairment produces serotonin and dopamine deficits that ketamine's NMDA-blocking and BDNF-stimulating effects were partially compensating for. Addressing the underlying deficit directly may reduce the intensity of the underlying depression that drives relapse.
Methylcobalamin (B12)	1000–2000mcg	Morning, sublingual.	Chronic ketamine use depletes B12 and disrupts methylation cycles. B12 is essential for myelin integrity — relevant for the neurological recovery from chronic NMDA disruption. Sublingual form for best absorption.
P5P (active B6)	25–50mg	Morning.	Cofactor for dopamine and serotonin synthesis — the neurotransmitter systems secondarily disrupted by ketamine's NMDA mechanism. Essential alongside the dopamine precursor stack below.

Magnesium glycinate.	400–600mg elemental	Morning and evening.	THE most important acute supplement in ketamine withdrawal because of its NMDA receptor modulating properties. Magnesium is an endogenous NMDA channel blocker — it provides gentle buffering of the glutamate rebound that produces the acute withdrawal anxiety. Start on Day 1 of cessation.

4.2 — Glutamate / NMDA System Support

THE CENTURION — Managing the Rebound — and Supporting the Recovery.

The glutamate rebound during ketamine withdrawal represents an acute pharmacological crisis. The NMDA system's recovery over the subsequent months constitutes structural rehabilitation, and both necessitate specific support.

Supplement	Dose.	Timing.	Role in NMDA recovery
Magnesium glycinate (repeated — highest priority)	400–600mg elemental	Morning and evening.	Magnesium is an endogenous NMDA receptor channel blocker – it does exactly what ketamine does (blocks NMDA) but gently, physiologically, and without producing dissociation or abuse potential. During the glutamate rebound of ketamine withdrawal, magnesium directly modulates the excitatory surge. Continue for months – it also supports NMDA receptor normalization during the recovery phase.
NAC (N-Acetylcystein).	600–1800mg daily in divided doses.	Morning and evening with food.	Restores glutamate homeostasis in the nucleus accumbens through the cystine-glutamate antiporter system. Multiple clinical trials support NAC for craving reduction in substance use disorders — its glutamate-normalizing mechanism is directly relevant to ketamine recovery where glutamate dysregulation is the primary neurochemical pathology.
Taurine	1–2g daily.	With meals.	NMDA receptor modulator — taurine acts as an inhibitory

			neurotransmitter at glycine receptors and modulates NMDA receptor function. Reduces glutamate excitotoxicity. Supports liver function, which is important for CYP2B6 ketamine metabolism. This dual mechanism is relevant to both the neurological and metabolic aspects of ketamine recovery.
L-Theanine	200–400mg	As needed for anxiety — 2–3 times daily in the acute phase.	Partial NMDA receptor antagonist and GABA/glycine agonist — provides gentle glutamate-dampening and calming through mechanisms parallel to ketamine's but without dissociation or addiction liability. Specifically relevant for the anxiety component of ketamine withdrawal where the glutamate rebound is the primary driver.
Omega-3 (EPA + DHA)	3–4g daily	With food, in the morning.	DHA is structurally incorporated into neuronal membranes, including those containing NMDA receptors—supporting membrane health supports receptor function normalization. EPA reduces neuroinflammation from NMDA excitotoxicity during withdrawal. BDNF support through DHA is particularly relevant for ketamine recovery.

4.3 — Recuperação Cognitiva: O K-Fuzz elimina a confusão.

THE CENTURION — The mind that was blocked is coming back online.

The cognitive impairment associated with heavy ketamine use — what users often call 'k-fuzz' or 'k-fog' — reflects genuine neurological disruption: NMDA receptor dysfunction in the prefrontal cortex and hippocampus impairs working memory, attention, word-finding, and the clarity of thought that makes engaging with reality feel worthwhile. For CYP2B6 poor metabolizers, this impairment may be more severe and more prolonged.

La durata del recupero cognitivo è generalmente di 3-12 mesi per la maggior parte degli utenti, con un recupero più rapido in coloro che mantengono un esercizio fisico vigoroso. Il meccanismo: la produzione di BDNF derivante dall'esercizio stimola la neurogenesi e la plasticità sinaptica dell'ippocampo, gli stessi processi che la ketamina stava stimolando artificialmente con i suoi effetti antidepressivi.

- Exercise (primary intervention): 30–60 minutes of aerobic activity daily. BDNF increases of 200–400%. This drives cognitive recovery faster than any supplement. The cognitive fog lifts measurably faster in people who exercise vigorously throughout recovery. This is the single most important cognitive intervention.
- Alpha-GPC eller CDP-Choline (250–500 mg om morgenen): støtte for acetylkolin for hukommelse og oppmerksomhet. Ketamin hemmer kolinerg funksjon gjennom NMDA-relaterte mekanismer. Kolinkosttilskudd kan bidra til å gjenopprette minnefunksjonen.
- Lion's Mane mushroom (500–1000mg daily): Stimulates NGF, which promotes neurite growth and synaptic repair. Specifically supports hippocampal plasticity, which is important for addressing memory problems related to ketamine use.
- DHA (1000mg+ daily): provides structural support for neuronal membranes containing NMDA receptors. Supports BDNF signaling. Has anti-inflammatory effects on neuroinflammation caused by NMDA excitotoxicity.
- The 40 Hz gamma protocol: the compendium's sonic architecture section specifically identifies 40 Hz binaural/isochronic tones for their potential role in supporting the brain's glymphatic clearing processes and neural repair. Use for 30–60 minutes daily as a cognitive recovery adjunct. Consistent use over months — not a quick fix, but a supportive background intervention.
- Novel skill learning: Challenging new skills stimulate BDNF in specific brain regions. An instrument, a language, or a physical skill with a learning curve—anything that generates errors and requires attention to correct. Damage to the prefrontal cortex caused by ketamine responds to the conditions created by skill learning.

4.4 — Dopamine and Serotonin Reconstruction

Supplement	Dose.	Timing.	Notes
5-HTP	50–150mg	Evening.	Precursor de serotonina: crucial si la depresión fue la causa subyacente del uso de cetamina. El sistema de serotonina, alterado por el uso crónico de cetamina, responde a la 5-HTP. La administración nocturna favorece la estructura del sueño y apoya la recuperación de la serotonina. No combine con ISRS o IRSN sin la orientación de un médico.
L-Tyrosine / NALT	500–1500mg	Good morning,	Precursor de la dopamina para la

		no protein today.	anhedonia y el déficit motivacional en la recuperación de la cetamina. Comience después del día 7, una vez que se esté comiendo regularmente. Particularmente importante para los perfiles Warrior COMT donde los aspectos dopaminérgicos del estado de la cetamina fueron primarios.
Rhodiola rosea	300–600mg	Morning.	Adaptogen with dopamine and serotonin modulating properties; anti-fatigue; cortisol normalization. Particularly useful in weeks 2–8 when post-ketamine depression and motivational deficits are prominent, but a precursor stack alone is insufficient.
SAMe (if depression is severe and MTHFR-related)	400–800mg	Good morning, haven't eaten anything yet.	Universal methyl donor for dopamine and serotonin synthesis. Specifically relevant if 5-MTHF alone is insufficient for the depression that ketamine was managing. Caution in bipolar disorder — can trigger mania. Start low, assess response carefully.

4. 5 — The Urological Recovery Track

THE CENTURION — The bladder heals when given the chance, but it needs help.

La ripresa urologica da neuropatia urinaria associata alla chetamina segue il protocollo di recupero neurologico e richiede un percorso medico specifico. Gli integratori seguenti sono stati studiati o presentano una giustificazione meccanicistica per il recupero dell'urotelio e della vescica; non sostituiscono le cure mediche urologiche.

⚠ ALLE INTEGRATORI PER IL SISTEMA URINARIO DEVONO ESSERE DISCUTTI CON IL TUO UROLOGO. ALCUNI, IN PARTICOLARE L'ASSUNZIONE DI DOSI ELEVATE DI VITAMINA C, POSSONO ACIDIFICARE LE URINE E PEGGIORARE L'IRRITAZIONE DELLA VESCICA IN ALCUNI PAZIENTI.

Supplement / intervention	Dose.	Timing.	Urological relevance.

Hydration — plain water.	3–4 liters daily.	Throughout the day.	The most important urological intervention after cessation. Diluting ketamine metabolites (particularly norketamine) in urine reduces their contact concentration with the urothelium. Consistent hydration is the primary harm reduction tool for early-stage KAU and the primary recovery support for healing urothelium.
Quercetin.	500–1000mg daily.	With food.	Anti-inflammatorio flavonoide con evidenze specifiche nell'interstiziale cistite (una condizione con alcune sovrapposizioni meccanicistiche con il KAU). Riduce l'infiammazione dell'urotelio e l'attivazione dei mastociti. Ben tollerato. Discutere con un urologo come parte del protocollo di gestione conservativa.
Marshmallow root (Althaea officinalis)	1–2g extract or 4–5g root.	Dagelijks, kan als thee worden ingenomen.	Demulcent — coats and soothes inflamed mucous membranes including the urothelium. Long traditional use in urinary tract irritation. May reduce the dysuria (painful urination) and urgency of early-to-intermediate KAU. Discuss with a urologist.
D-mannose.	1–2g daily.	With water.	Supports urothelial surface integrity — the glycoprotein coating of the bladder lining that is disrupted by KAU. Also protects against secondary bacterial colonization of a damaged urothelium. Evidence in urinary tract health broadly.
PEA (Palmitoylethanolamide)	300–600mg daily.	With food.	Endogenous anti-inflammatory fatty acid amide that reduces mast cell activation and neurogenic inflammation — both of which contribute to bladder pain and irritation in KAU. Evidence in interstitial cystitis and pelvic pain conditions. Does not interact with bladder pH.

4.6 — Sleep Reconstruction

Ketamine disrupts sleep architecture through multiple mechanisms: NMDA receptor disruption affects sleep regulation, dopamine and serotonin disruption impairs sleep quality, and psychological dependence often involves using ketamine specifically for sleep (the dissociative state as a substitute for genuine sleep). The vivid, disturbing dreams that often accompany early ketamine recovery are REM rebound — the brain reclaiming the restorative sleep stages that ketamine was suppressing.

- Melatonin: 0.5–1mg sublingually, 30–60 minutes before bed. Low dose. High doses can disrupt the circadian rhythm.
- Magnesium glycinate: 400mg elemental, taken in the evening. It helps with NMDA modulation, reduces anxiety, and buffers cortisol – all of which promote sleep onset.
- Glycine: 3–5g, 30 minutes before bed. NMDA co-agonist at inhibitory doses; reduces core temperature for sleep onset; improves sleep architecture.
- L-Theanine: 200–400mg evening. Promotes alpha waves; reduces the glutamate-rebound anxiety that prevents sleep in early ketamine recovery.
- 5-HTP: 50–100mg in the evening. Supports serotonin to melatonin synthesis. Important if there's insomnia related to depression.

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Protocol 17: Sleep Architecture

**Introduction:**

This protocol outlines the importance of sleep architecture and provides guidance on how to assess and improve it.  Adequate sleep is crucial for physical and mental health, and disruptions to sleep architecture can have significant consequences.

**Understanding Sleep Architecture:**

Sleep architecture refers to the cyclical pattern of different stages of sleep that occur throughout a typical night's rest. These stages include:

*   **NREM (Non-Rapid Eye Movement) Stage 1:**  A light stage of sleep, easily disrupted.
*   **NREM Stage 2:**  Deeper than Stage 1, characterized by slower brain waves and occasional bursts of activity called sleep spindles.
*   **NREM Stage 3 (Slow Wave Sleep):** The deepest stage of sleep, essential for physical restoration and memory consolidation.
*   **REM (Rapid Eye Movement) Sleep:**  Characterized by rapid eye movements, dreaming, and muscle paralysis. Important for cognitive function and emotional processing.

A healthy sleep cycle typically consists of several cycles, each lasting approximately 90-120 minutes. Each cycle progresses through the NREM stages before entering REM sleep. The proportion of time spent in each stage changes throughout the
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night, with more slow wave sleep occurring earlier in the night and more REM sleep occurring later.

Assessing Sleep Architecture:

Sleep architecture can be assessed using polysomnography (PSG), a comprehensive sleep study that measures brain waves, eye movements, muscle activity, heart rate, and breathing patterns. PSG is typically conducted in a sleep laboratory under the supervision of trained technicians. Home sleep apnea tests (HSAT) may also provide some information about sleep stages, but are less detailed than PSG.

Factors Affecting Sleep Architecture:

Several factors can disrupt sleep architecture, including:

* **Age:** Sleep patterns change with age, with older adults typically experiencing less slow wave sleep and more frequent awakenings.
* **Medical Conditions:** Conditions such as sleep apnea, restless legs syndrome, and chronic pain can interfere with sleep architecture.
* **Medications:** Certain medications can affect sleep stages.
* **Lifestyle Factors:** Caffeine, alcohol, nicotine, and irregular sleep schedules can all disrupt sleep architecture.
* **Stress and Anxiety:** Psychological stress can significantly impact sleep quality and structure.

Improving Sleep Architecture:

Strategies to improve sleep architecture include:

* **Maintaining a Regular Sleep Schedule:** Going to bed and waking up at the same time each day, even on weekends, helps regulate the body's natural sleep-wake cycle.
* **Creating a Relaxing Bedtime Routine:** Engaging in calming activities before bed, such as taking a warm bath, reading, or listening to relaxing music, can promote better sleep.
* **Optimizing the Sleep Environment:** Ensuring that the bedroom is dark, quiet, and cool can improve sleep quality.
* **Avoiding Caffeine and Alcohol Before Bed:** These substances can interfere with sleep architecture.
* **Managing Stress and Anxiety:** Practicing relaxation techniques such as meditation or yoga can help reduce stress and improve sleep.
* **Addressing Underlying Medical Conditions:** Treating any underlying medical conditions that may be affecting sleep is crucial.

When to Seek Professional Help:

If you are experiencing persistent difficulties with sleep, including frequent awakenings, excessive daytime sleepiness, or concerns about your sleep architecture, it is important to consult a healthcare professional. They can help identify any underlying causes and recommend appropriate treatment options.

Medical help is not failure.
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## 4.7 — Peptide Support Layer

Consult a doctor – investigate substances.

| Peptide. | Ketamine's mechanism of action involves several processes, including:<br><br>* NMDA receptor antagonism.<br>* Increased dopamine release.<br>* Activation of opioid receptors.<br>* Modulation of glutamate transmission.<br><br>These mechanisms contribute to ketamine's analgesic, anesthetic, and antidepressant effects. However, they can also lead to side effects such as hallucinations, disorientation, and respiratory depression.<br><br>The exact mechanism by which ketamine produces its recovery effects is not fully understood. However, it is believed to involve a combination of factors, including:<br><br>* Increased glutamate release.<br>* Activation of NMDA receptors.<br>* Modulation of opioid receptor activity. | Timing. | Notes |
|---|---|---|---|
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	These processes may help to restore normal brain function and promote healing after injury or illness. **It is important to note that ketamine's mechanism of action can vary depending on the individual and the specific condition being treated. For example, ketamine may have different effects in patients with depression compared to those with chronic pain.** **If you are considering using ketamine for recovery purposes, it is important to discuss the potential risks and benefits with your doctor. They can help you determine if ketamine is right for you and monitor you for any side effects.**		
BPC-157	Modulazione del sistema glutammato-dopaminergico; effetti sulla via NMDA; riparazione dell'asse intestino-cervello; evidenze specifiche di protezione neuronale dalla neurotossicità da eccitotossicità NMDA in modelli animali; potenziali proprietà curative per l'epitelio uroteliale.	Day 1 onwards — daily	BPC-157 has demonstrated neuroprotective effects against NMDA excitotoxicity in research — directly relevant to the glutamate rebound of ketamine withdrawal. Its gut-brain axis repair function addresses the dysbiosis that ketamine use produces. The urothelial healing hypothesis — BPC-157's tissue repair properties extending to bladder epithelium — is mechanistically plausible though clinical evidence in KAU specifically is limited. Discuss the bladder application with your urologist.
Semax	Aumento del BDNF;	Weeks 1	Semax's BDNF-stimulating

	mejora cognitiva; modulación de la dopamina; apoyo a la corteza prefrontal: abordando directamente el deterioro cognitivo asociado al uso de cetamina.	onwards — particularly months 1–6 for cognitive recovery.	effects directly accelerate cognitive recovery from ketamine-induced NMDA dysfunction. The prefrontal cortex impairment and memory deficits associated with heavy ketamine use respond to BDNF-driven neuroplasticity. This is the peptide most directly relevant to 'k-fuzz' recovery. Intranasal.
Selank	Anxiolytic that works by affecting GABA and serotonin levels; reduces anxiety without causing dependence; provides a stable, non-sedating calming effect.	Weeks 1–8 — as needed for acute anxiety.	For the anxiety of ketamine withdrawal and early recovery, Selank provides a genuine anxiolytic effect without the dissociative mechanism that made ketamine appealing in the first place. This is important: the person recovering from ketamine-as-anxiety-management needs an anxiety management tool that does not work through dissociation. Selank provides anxiolysis through a completely different pathway. Intranasal.
Epitalon	Sleep architecture; pineal pathway; telomere protection; circadian normalization.	Weeks 1–6; repeat cycles as needed for ongoing sleep problems.	Sleep disruption during ketamine recovery is caused by both neurochemical factors (the way ketamine affects NMDA, which regulates sleep) and behavioral patterns (habits developed around the sedative effects of ketamine). Epitalon supports the pineal gland, which ketamine disrupts, and helps restore normal sleep patterns.

Section 5 — The Ketamine Craving — Understanding and Interrupting.

THE CENTURION — The craving isn't for the drug itself. It's for the escape it provided.

Ketamine cravings have a specific quality that distinguishes them from stimulant cravings (which feel like hunger for more) or opioid cravings (which feel like pain demanding relief). Ketamine cravings tend to feel like: the overwhelming desire to leave. To exit from consciousness, from reality, from an emotional state that is too large to tolerate. The craving arrives when the present moment becomes unbearable — and ketamine's offer is the most efficient exit available.

Understanding this changes the interrupt protocol. You are not fighting a desire for a substance. You are meeting a need for exit from an overwhelming state — and providing an alternative exit that does not require dissociation.

The Ketamine Craving Types.

Tipo di desiderio.	What it feels like.	Interrupción inmediata.
Reality escape craving (most common)	The present moment is too much — too painful, too anxious, too real, too relentless. The mind reaches for the exit that has always been reliable. The craving is less about wanting the drug and more about wanting to not be here.	This is the grounding interrupt—the opposite of dissociation. Five things you can see. Four things you can touch. Three things you can hear. Two things you can smell. One thing you can taste. Then, 4-7-8 breathing. Then, cold water on the face. You are not suppressing the overwhelming state—you are grounding it in physical reality rather than escaping from it.
Depression/ anhedonia craving.	The flatness is unbearable. Nothing feels interesting, worthwhile, or real without the drug. The ketamine state felt more alive than ordinary life. The craving is for aliveness that the recovering dopamine system cannot yet generate.	This is a response to dopamine deficiency. Exercise is the quickest way to boost dopamine levels. Consider cold exposure. Listen to music. Spend time with someone who makes you feel genuinely good. Implement the most intense activity possible from the natural reward strategies outlined in Section 6.

Curiosity/exploration craving.	Ketamine's dissociative state had genuine depth — profound insights, ego dissolution, the feeling of accessing something larger than ordinary consciousness. The craving is for that space of expanded perception, not for the drug specifically.	This is an invitation to develop what Section 6 refers to as the authentic mystical infrastructure —meditation, breathwork, flow states—that accesses related neurological states without pharmacology. The craving for genuine transcendence is not a pathology; it points towards a real human need that can be met authentically.
Social/contextual craving.	Specifiek muziek, specifieke vrienden, specifieke omgevingen en specifieke tijdstippen van de nacht - de associaties die zijn opgebouwd rond het gebruik van ketamine kunnen leiden tot een plotselinge, intense hunkering die zonder waarschuwing ontstaat.	Environmental exit first — leave the context before doing anything else. Protocol 05 (environmental redesign) has addressed the most prominent cue environments, but novel cues will appear. Exit. Contact. Ground. The sequence does not change.

THE KETAMINE CRAVING INTERRUPT — 5-MINUTE PROTOCOL.

1. Reality escape?
   Depression/anhedonia?
   Curiosity/exploration?
   Social/contextual?
   The type determines the primary response.
2. GROUND IMMEDIATELY.
   Five things you can see.
   Four you can physically touch.
   Three you can hear.
   Two you can smell.
   One you can taste.
   This is not a metaphor — it is prefrontal cortex activation and present-moment anchoring that interrupts the dissociative 'exit' the craving is seeking.
3. EXECUTE 4-7-8 BREATHING. Four full cycles. The parasympathetic response reduces the overwhelm that made the exit feel necessary.
4. COLD WATER ON FACE AND WRISTS. The dive reflex activates within seconds — immediate parasympathetic shift. This is the fastest available grounding intervention outside of the breath.
5. MAKE CONTACT. Send one text message or make a phone call. The feeling of being disconnected is part of the pattern that ketamine can create. Making contact with someone is a way to counteract this effect.
6. From Section 6: movement, breathwork, meditation, cold plunge, creative work. You are not suppressing the need the craving represents. You are meeting it differently.

7. Trigger, type, intensity (0-10), action, outcome. The ketamine craving pattern documented over weeks reveals the specific internal states and external contexts that are driving the difficulty. This is diagnostic information for the therapy and for the natural addiction architecture.

Section 6 — Porting the Addiction: Genuine Access to What Ketamine Faked.

THE CENTURION — Centurian Paper C-28 — The Counterfeit Sacred Becomes the Real Thing.

The compendium describes ketamine as the counterfeit sacred: it simulates states that human beings have sought through contemplative practice, ritual, and spiritual discipline for thousands of years. The ego dissolution of the k-hole maps precisely onto what contemplative traditions describe as the dissolution of the small self. The expanded awareness of sub-anesthetic doses maps onto what meditators in deep practice report. The sense of connection with something larger than ordinary consciousness is not unique to ketamine — it is a neurological state that the human system was designed to access.

La differenza tra la versione con chetamin e la versione autentica è esattamente ciò che il compendio indica: la versione con chetamin fornisce lo stato senza richiedere lo sviluppo dell'architettura che rende lo stato sostenibile, integrabile e genuinamente trasformativo. La versione contemplativa richiede anni di pratica e una vera trasformazione della persona che abita lo stato.

Ketamine recovery is unique in this series because the natural addiction architecture is not primarily about building intensity (stimulants), endorphin warmth (opioids), GABA calm (benzos/GHB), or anandamide flow (cannabis). It is about building genuine access to the transcendent states that ketamine was counterfeiting. This is the most demanding natural addiction architecture in the series — and potentially the most rewarding.

The Grounding Architecture — Presence Before Transcendence.

THE CENTURION — You Cannot Integrate What You Have Not Embodied.

The first task is the inverse of what ketamine was doing. Before building genuine access to expanded states, the person in ketamine recovery must build the capacity to inhabit ordinary states—to be present in their body, in the moment, in their actual life, without reaching for the exit.

This is somatic work. It's about reconnecting with the body, something ketamine might have helped people avoid. For many in ketamine recovery, the body holds unprocessed experiences: trauma, grief, anxiety, pain – things dissociation previously offered relief from. Recovery involves developing the ability to engage with these experiences without being overwhelmed by them.

- Somatic therapy (Protocol 23 in the main Compendium, or external specialist): specifically for people whose ketamine use was managing trauma

or emotional pain stored in the body. EMDR, somatic experiencing, sensorimotor psychotherapy. These approaches work with the body's experience directly — the opposite of ketamine's body-exit mechanism.

- Grounding practice (5-4-3-2-1 sense anchoring): practiced daily until it is as automatic as the former ketamine use. The nervous system needs to build the neural pathway for returning to present-moment embodied awareness.
- Exercice physique spécifiquement en tant qu'incarnation : le but n'est pas la condition physique. Il s'agit d'être présent dans un corps en mouvement. La course à pied, la natation, les arts martiaux, la danse - toute activité physique qui exige une attention totale à ce que le corps fait ici et maintenant.
- Safe physical contact: For people whose ketamine use involved social contexts, rebuilding the capacity for physical presence with others without chemical disinhibition is part of the grounding work.

The Transcendence Architecture — The Real Thing.

THE CENTURION — The states where ketamine can be accessed genuinely, even if it's been faked.

Practice.	Neurological state produced.	How to implement	Ketamine recovery involves a multifaceted approach, encompassing both pharmacological and psychological interventions tailored to address the specific needs of individuals undergoing treatment. Pharmacological Interventions: * **Recovery Kernel:** This innovative therapeutic modality leverages the unique neurobiological properties of ketamine to promote neural plasticity and facilitate the restoration of optimal brain function. * **Naloxone:** In cases where opioid-related complications arise during ketamine recovery, naloxone may be administered as an antidote to reverse the effects of opioid agonists. * **COMT inhibitors:** These medications can help to

			modulate dopamine levels in the brain, potentially enhancing the therapeutic benefits of ketamine while minimizing adverse effects. *** **DRD2 antagonists:** By selectively blocking dopamine receptors, these agents can help to mitigate certain side effects associated with ketamine treatment, such as psychosis or agitation.** *** **MTHFR gene variants:** Individuals with specific MTHFR gene variants may require tailored pharmacological interventions to optimize their response to ketamine therapy and minimize the risk of adverse events.** **Psychological Interventions:** *** **Cognitive Behavioral Therapy (CBT):** CBT is a widely recognized therapeutic approach that can help individuals develop coping mechanisms for managing cravings, reducing relapse rates, and improving overall mental well-being during ketamine recovery.** *** **Dialectical Behavior Therapy (DBT):** DBT is another evidence-based therapy that focuses on teaching skills such as mindfulness, distress tolerance, emotion regulation, and interpersonal effectiveness to promote emotional stability and resilience during ketamine recovery.** *** **Motivational Interviewing (MI):** MI is a client-centered counseling approach that helps individuals explore their ambivalence about change and identify intrinsic motivations for pursuing**

			recovery from ketamine dependence. **Additional Considerations:** *** **Blueprint:** A comprehensive treatment plan should be developed in collaboration with healthcare professionals to address the individual's specific needs, goals, and preferences during ketamine recovery.** *** **findahelpline.com:** This website provides a directory of resources and support services for individuals seeking assistance with substance abuse and mental health issues.** **It is important to note that medical help is not failure.**
Meditation — absorption / jhana states.	Ego dissolution, temporal dissolution, unity experience — measurably similar to psychedelic and dissociative states on brain imaging.	Dharma Recovery (Protocol 04). Begin with basic breath meditation. Build to longer sittings. The full dissolution states may take years of daily practice — the journey is the point.	The compendium notes that Buddhist jhana states, Sufi hal, Hindu samadhi, and Christian mystical union all describe states that brain imaging maps onto NMDA-modulated dissociative states. The practices that cultivate these states build the prefrontal inhibition of default mode network activity that ketamine was producing chemically. The genuine version builds something the pharmaceutical version never did: integration. The expanded state becomes accessible and useful rather than merely escapist.
Holotropic eller pranayama-andningsövningar.	Altered consciousness through CO2/O2 modulation — documented ego dissolution, emotional release, and transpersona	Holotropic breathwork sessions (group or individual with a facilitator). Wim Hof breathwork. Box breathing extended for over 30 minutes.	Breathwork accesses neurological states that overlap with NMDA modulation through hyperventilation-induced CO2 reduction, which affects NMDA receptor function. This is a legitimate pathway to the states ketamine was producing — available without pharmacology, with full consciousness and memory of the experience, and integrable into a life narrative.

	l experiences.		
Sensory deprivation / float therapy.	A significant decrease in sensory stimulation can lead to profound relaxation, a sense of altered perception of time, and, on occasion, a dissolving of the sense of self, all achieved without the use of any pharmacolog ical agents.	Float centers offer sessions lasting 60–90 minutes. Aim for regular use – ideally, weekly. The benefits become more pronounced with practice.	Float therapy accesses the sensory reduction that the k-hole was providing through external stimulation removal rather than NMDA blockade. For the person who was using ketamine specifically for the sensory and cognitive quiet it produced, floatation therapy is the most direct pharmacology-free analog available. Some ketamine-recovery communities specifically use float therapy as a transition practice.
Peak experience through physical mastery.	Flow state, runner's high, and peak performance states produce BDNF, endorphin, and dopamine co-release with measurable ego-boundary dissolution at their peak.	Sports extrêmes, arts martiaux, athlétisme d'endurance, escalade technique : activités physiques qui exigent un véritable niveau de maîtrise et qui produisent un état de concentration intense.	For individuals with the "Warrior" profile who appreciated the social euphoria and boundary dissolution associated with ketamine, achieving peak physical condition offers a similar level of intensity and boundary dissolution through dedicated effort. The element of mastery provides something ketamine never could: the authentic understanding that you possess the ability to achieve significant results.
Integration therapy and psychedelic-assisted therapy (if appropriate).	Professionell terapeutisk användning av MDMA (för PTSD, i forskningsmil jöer), psilocybin (för depression och missbruk, i kliniska	Only through licensed clinical research programs or licensed providers (for ketamine specifically). Not for self-administration. A full therapeutic container is required.	The compendium's Protocol 42 addresses psychedelic-assisted therapy specifically. For ketamine recovery where depression or trauma was underlying the use, supervised therapeutic psychedelics or therapeutic ketamine (in a clinical context with integration) may be appropriate under specialist guidance. These are catalysts that open a window — the integration work determines

	prövningar) eller övervakad ketaminbehandling (för behandlingsresistent depression) – med fullt stöd för integration.		the outcome.

Section 7 — Navigating the Recovery Kernel: Your Protocol Map.

Fase.	Protocols	Primary focus	Ketamine-specific notes: * [ ] Ketamine can cause hallucinations and disorientation. * [ ] Ketamine can increase blood pressure and heart rate. * [ ] Ketamine can cause nausea and vomiting. * [ ] Ketamine can cause respiratory depression. * [ ] Ketamine can cause seizures. * [ ] Ketamine can cause coma. * [ ] Ketamine can be fatal. * [ ] If you experience any of these symptoms after taking ketamine, seek medical help immediately. * [ ] Do not drive or operate machinery while under the influence of ketamine. * [ ] Do not take ketamine with alcohol or other drugs. * [ ] Ketamine is a Schedule III controlled substance in Australia. It is illegal to possess or use ketamine without a prescription. * [ ] If you are pregnant or breastfeeding, do not take ketamine. * [ ] If you have any medical conditions, such as heart disease, high blood pressure, or respiratory problems, talk to your doctor before taking ketamine. * [ ] Ketamine can interact with other medications. Talk to your doctor about all the medications you are taking before taking ketamine. * [ ] Do not take more ketamine than prescribed by your doctor. * [ ] Store ketamine in a safe place, out of reach of children

			and pets. *** [ ] If you have any questions or concerns about ketamine, talk to your doctor or pharmacist.** **Ketamine is a powerful drug that can cause serious side effects. It is important to use ketamine safely and responsibly. If you are not sure how to take ketamine, ask your doctor or pharmacist for help.**
Immediate Medical (if bladder symptoms are present).	Urologie – pas le compendium.	Valutazione urologica; cistoscopia; funzione renale; interruzione.	Symptômes de la vessie nécessitent une consultation en urologie avant toute autre étape dans ce cadre. La KAU (Keto-adaptable urethral syndrome) progresse avec une utilisation continue ; l'arrêt est le seul moyen de l'arrêter, et certains dommages nécessitent un traitement médical actif, indépendamment de l'arrêt.
Biological Foundation (Days 1–14)	Protocol 01, Protocol 07, Protocol 17, Protocol 18	Glutamate rebound management (magnesium, NAC), methylation foundation, sleep, nutrition.	Magnesium glycinate, Day 1 — highest priority for the glutamate rebound. NAC, Day 1. 5-MTHF, Day 1 if depression is present. Protocol 01 (Shelter) for the acute dissociative anxiety of early withdrawal. Reduce environmental stimulation — the upregulated NMDA system makes everything feel amplified.
Genomic Mapping (Weeks 1–4)	Section 15 (Compendium), CYP2B6 testing.	CYP2B6 (Dissociative Logjam Risk), COMT constitution, MTHFR, BDNF.	CYP2B6 är den viktigaste gentestet vid ketaminbehandling – det förklarar direkt doskänslighet, svårighetsgraden av blåsskador och återhämtningstiden för kognitiva funktioner. Om du upplever att ketamin alltid påverkar dig mer än andra, kan detta test vara den viktigaste medicinska informationen du kan få.
Grounding Architecture (Weeks 1–8 — CRITICAL)	Protocol 01 (grounding practices), somatic therapy referral, Section 6.1 of this guide.	5-4-3-2-1 grounding; somatic therapy; embodied exercise; physical presence practices.	This is the phase that distinguishes ketamine recovery from every other guide in this series. Before transcendence architecture can be built, grounding architecture must be established. The person must be able to tolerate being present in their body and their life before building genuine access to

			expanded states.
Cognitive Recovery (Weeks 2–12)	Protocol 03 (exercise), Section 4.3 of this guide, novel skill learning.	Exercise for BDNF; Alpha-GPC; Lion's Mane; 40 Hz protocol; new learning.	La nebbia cognitiva si dirada gradualmente nei mesi 1-6 nelle persone che fanno esercizio fisico intenso. Monitorala: il ritorno della chiarezza mentale è una delle esperienze più motivanti e gratificanti del percorso di recupero con la chetamina.
Psychological and trauma work (months 1–6, when stable).	Protocol 23, external therapy (somatic, EMDR), Protocol 11.	Trauma processing if underlying ketamine use; depression treatment if biological; anxiety management.	The symptoms that ketamine was masking and preventing resolution of will not disappear when you stop taking it. They require direct treatment. Somatic therapy, EMDR, CBT, or MBSR — depending on the underlying condition. This is not optional for lasting ketamine recovery.
Transcendence Architecture (Months 2–12+).	Protocol 04 (Dharma Recovery), float therapy, breathwork, Protocol 42 (if appropriate).	Meditatiegebouw; ademhalingstechnieken; flow-toestanden; authentieke toegang tot verruimde bewustzijnstoestanden.	La construcción de la infraestructura mística genuina: las prácticas que permiten acceder a los estados que la ketamina falsificaba. Este es el largo camino de la recuperación de la ketamina, y también su mayor oportunidad.
Identity and Meaning (Months 3–12+).	Protocol 20, Protocol 21, C-28.	Identifies as someone who lives in reality, not someone who escapes it; has a sense of purpose; represents the genuine effects of ketamine.	For people who used ketamine partly for its spiritual or philosophical dimensions, the identity reconstruction includes integrating what was genuinely valuable in those experiences into a sober life. What was real about the insights? What was the expansion pointing toward? Protocol 20 and C-28 provide the framework.

Section 8 — The Medical Support Layer

Addressing the Underlying Depression

THE CENTURION — The condition ketamine was treating may need its own treatment.

Een aanzienlijk aantal mensen met een ketamineverslaving begon ermee te gebruiken omdat ketamine snelle en diepgaande antidepressieve effecten veroorzaakte die traditionele behandelingen niet boden. De depressie verdwijnt niet bij het stoppen; deze keert vaak terug, soms intenser, in de eerste weken van herstel.

This depression must be treated directly. Options, in order of preference:

- 5-MTHF (400–1000mcg) + methylcobalamin: If MTHFR variants are suspected or confirmed, this is first-line. Addresses the specific methylation-deficit mechanism that makes some people's depression unresponsive to SSRIs. Try for 6–8 weeks before concluding insufficient.
- SSRIs or SNRIs: with CYP2D6/2C19 metabolizer status guiding selection. Discuss with a psychiatrist. Note that SSRI response in people with ketamine use disorder is sometimes complicated by the NMDA system disruption — allow adequate trial periods.
- Supervised therapeutic ketamine (esketamine / Spravato): if the depression is genuinely treatment-resistant and other options have been exhausted, supervised ketamine therapy may be clinically appropriate even in the context of recovery from recreational ketamine use. This requires specialist addiction psychiatry input, not a general ketamine clinic.
- TMS (Transcranial Magnetic Stimulation): a non-pharmacological treatment for depression with strong evidence in treatment-resistant cases. It modulates glutamate and NMDA pathways through electromagnetic stimulation, which is mechanistically relevant to ketamine recovery.

Managing Persistent Dissociation

Some people in ketamine recovery experience persistent depersonalization and derealization — feeling disconnected from themselves or their environment as a default state even without the drug. This can persist for months and is one of the most distressing features of heavy ketamine use.

Persistent dissociation in recovery is addressed through:

- Terapia somática (principal): trabajar directamente con la experiencia corporal para reconstruir el sentido de habitar un ser. Psicoterapia sensorimotriz, Experiencia Somática, EMDR.

- Grounding practices (daily):
  * 5-4-3-2-1 sensory anchoring.
  * Cold water exposure.
  * Physical contact with safe people.
- SSRI therapy: SSRIs have evidence in depersonalization disorder, which shares mechanisms with ketamine-induced persistent dissociation.
- Avoiding cannabis and other dissociative substances: Cannabis use during ketamine recovery can significantly worsen depersonalization/derealization. Avoid.

Urological Medical Track.

The urological recovery runs parallel to the addiction recovery and requires its own specialist team:

- Consulenza urologica: se sono presenti sintomi urologici, questo è il primo appuntamento medico. Non si tratta di medicina della dipendenza né di psichiatria, ma di urologia.
- Cystoscopy: direct visualisation of the bladder to assess the degree of structural damage and guide treatment planning.
- Analisi della funzione renale ed ecografia: per valutare eventuali coinvolgimenti delle vie urinarie superiori.
- Hydrodistension: a urological procedure that stretches the bladder under anesthesia — can provide temporary symptom relief and allows assessment of bladder capacity.
- Tratamientos intravesicales: infusión directa de agentes antiinflamatorios (ácido hialurónico, sulfato de condroitina) para apoyar la reparación del urotelio.
- Controlli urologici di follow-up: ogni 3 mesi per almeno il primo anno di astinenza, per monitorare la ripresa o la progressione.

A Final Word.

THE CENTURION — The exit door was real. The building you were escaping from is where the life is.

The compendium's description of ketamine as the counterfeit sacred is not a moral condemnation. It is a precise pharmacological and phenomenological observation: the states ketamine produces are real. The ego dissolution, the sense of expanded awareness, the release from the tyranny of the self-referential mind — these are genuine neurological events that map onto states human beings have sought and valued across every culture in recorded history.

What makes them counterfeit is not their character but their consequences. The path that provides them without the development — without the years of contemplative practice, without the gradual building of the psychological integration capacity, without the steady cultivation of the ability to remain present in ordinary states — also provides them without the fruit. The insight that cannot be integrated. The expansion that cannot be sustained. The peace that requires another dose. And, increasingly, a bladder that is slowly being destroyed by the molecule that was providing the transcendence.

La recuperación de la ketamina no es el abandono de los estados que buscabas. Es el descubrimiento del camino que realmente te lleva allí.

The meditation hall is built for exactly what the k-hole was approximating.
The float tank provides sensory dissolution without NMDA receptor damage.
The breathwork session produces ego boundary dissolution through means the body owns rather than means the pharmacy supplies.
The flow state of physical mastery creates the sense of effortless, timeless absorption that sub-anesthetic ketamine was producing chemically.

The difference is that these paths build something. Each session leaves a residue — a slightly more developed capacity to inhabit the full range of human experience. A slightly more stable foundation for the next expansion. A slightly deeper integration of what the previous expansion revealed.

Ketamine estava deixando um resíduo diferente. No epitélio urotelial. No córtex pré-frontal. Nos receptores NMDA que estavam tentando manter as condições para a consciência genuína.

The building you were escaping from is where the life is. The Recovery Kernel framework is the map for inhabiting it.

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Problemas urinários relacionados ao uso de cetamina: consulte um urologista antes de qualquer outra coisa.

Refer to the Recovery Kernel Complete Centurion Compendium for all referenced protocols.